What is claimed is:

1. A method of preventing or treating an amyloid-β related disease in a subject, said method comprising administering to a subject in need thereof an effective amount of a first agent that prevents or treats amyloid-β related disease, and a second agent that is (i) a peptide or peptidomimetic that modulates amyloid-β fibril formation or induces a prophylactic or therapeutic immune response against amyloid-β fibril formation, or (ii) an immune system modulator that prevents or inhibits amyloid-β fibril formation.

2. The method of claim 1, wherein said first agent prevents or inhibits amyloid-β fibril formation, neurodegeneration, or cellular toxicity.

3. The method of claim 1, wherein said amyloid-β related disease is Alzheimer's disease, mild cognitive impairment, mild-to-moderate cognitive impairment, vascular dementia, cerebral amyloid angiopathy, hereditary cerebral hemorrhage, senile dementia, Down's syndrome, inclusion body myositis, age-related macular degeneration, or a condition associated with Alzheimer's disease.

4. The method of claim 3, wherein said Alzheimer's disease is sporadic (non-hereditary) or familial (hereditary).

5. The method of claim 3, wherein said condition associated with Alzheimer's disease is selected from the group consisting of hypothyroidism, cerebrovascular disease, cardiovascular disease, memory loss, anxiety, a behavioral dysfunction, a neurological condition, or a psychological condition.

6. The method of claim 5, wherein said behavioral dysfunction is apathy, aggression, or incontinence.

7. The method of claim 5, wherein said neurological condition is Huntington's disease, amyotrophic lateral sclerosis, acquired immunodeficiency, Parkinson's disease, aphasia, apraxia,

agnosia, Pick disease, dementia with Lewy bodies, altered muscle tone, seizures, sensory loss, visual field deficits, incoordination, gait disturbance, transient ischemic attack or stroke, transient alertness, attention deficit, frequent falls, syncope, neuroleptic sensitivity, normal pressure hydrocephalus, subdural hematoma, brain tumor, posttraumatic brain injury, or posthypoxic damage.

8. The method of claim 5, wherein said psychological condition is depression, delusions, illusions, hallucinations, sexual disorders, weight loss, psychosis, a sleep disturbance, insomnia, behavioral disinhibition, poor insight, suicidal ideation, depressed mood, irritability, anhedonia, social withdrawal, or excessive guilt.

9. The method of claim 1, wherein said subject has a genomic mutation in an amyloid precursor protein gene, an ApoE gene, or a presenilin gene.

10. The method of claim 1, wherein said subject has amyloid-β deposits.

11. The method of claim 1, wherein said subject is a human.

12. The method of claim 1, wherein said amyloid-β is an amyloidogenic peptide produced from β-amyloid precursor protein.

13. The method of claim 12, wherein said β-amyloid is a peptide having 39-43 amino acids.

14. The method of claim 1, wherein said first agent prevents or inhibits β-amyloid fibril formation; prevents β-amyloid peptide, in its soluble, oligomeric form, or in its fibrillar form, from binding or adhering to a cell surface and causing cell damage or toxicity; blocks amyloid-induced cellular toxicity or microglial activation; blocks amyloid-induced neurotoxicity; reduces the rate or amount of β-amyloid aggregation, fibril formation, or deposition; slows the rate of amyloid-β fibril formation or deposition; lessens the degree of amyloid-β deposition; inhibits, reduces, or prevents amyloid-β fibril formation; inhibits amyloid-β induced

inflammation; enhances the clearance of amyloid-β from the brain; alters the equilibrium of amyloid-β between the cerebrospinal fluid or brain and the plasma and decreases the amount of amyloid-β in the brain versus the equilibrium distribution in an untreated subject; reverses or favors deposition of amyloid in a subject having amyloid deposits; favors plaque clearance or slows deposition in a subject having amyloid deposits; decreases the amyloid-β concentration in the brain of a subject versus an untreated subject; penetrates into the brain; maintains soluble amyloid in a non-fibrillar form; increases the rate of clearance of soluble amyloid from the brain of a subject versus an untreated subject; or inhibits or reduces an interation between amyloid-β and a cell surface constituent.

15. The method of claim 1, wherein said first agent is a substituted or unsubstituted alkylsulfonic acid, a substituted or unsubstituted alkylsulfuric acid, a substituted or unsubstituted alkylthiosulfonic acid, a substituted or unsubstituted alkylthiosulfuric acid, a substituted or unsubstituted lower alkylsulfonic acid, a (substituted- or unsubstituted-amino)-substituted alkylsulfonic acid, a (substituted- or unsubstituted-amino)-substituted lower alkylsulfonic acid, a substituted or unsubstituted straight-chain alkylsulfonic acid, a substituted or unsubstituted cycloalkylsulfonic acid, a substituted or unsubstituted branched-chain alkylsulfonic acid, or an ester or amide thereof, including pharmaceutically acceptable salts thereof.

16. The method of claim 15, wherein said amino substituent has the formula $-NR^aR^b$, wherein R^a and R^b are each independently hydrogen, an alkyl group, an aryl group, or a heterocyclyl group, or R^a and R^b, taken together with the nitrogen atom to which they are attached, form a heterocyclic moiety having from 3 to 8 atoms in the ring.

17. The method of claim 16, wherein said heterocyclic moiety comprises a piperidinyl or pyrrolidinyl group.

18. The method of claim 15, wherein said amino substituent comprises an alkylamino or dialkylamino group.

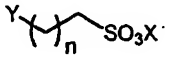
19. The method of claim 15, wherein said alkylsulfonic acid comprises an alkyl group substituted with at least a group of the formula $-SO_3H$ or $-SO_3^-X^+$, where X^+ is a cationic group at physiologic pH.

20. The method of claim 19, wherein said cationic group is a hydrogen atom, a sodium atom, or an amino group.

21. The method of claim 15, wherein said alkylsulfonic acid is substituted with a straight or branched alkyl or cycloalkyl group, or a group of the formula $-NH_2$, $-SO_3H$, $-OSO_3H$, $-CN$, $-NO_2$, $-F$, $-Cl$, $-Br$, $-I$, $-CH_2OCH_3$, $-OCH_3$, $-SH$, $-SCH_3$, $-OH$, or $-CO_2H$.

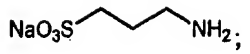
22. The method of claim 15, wherein said alkylsulfonic acid is substituted with a substituent selected from the group consisting of halogeno, trifluoromethyl, nitro, cyano, C_1-C_6 alkyl, C_2-C_6 alkenyl, C_2-C_6 alkynyl, C_1-C_6 alkylcarbonyloxy, arylcarbonyloxy, C_1-C_6 alkoxycarbonyloxy, aryloxycarbonyloxy, C_1-C_6 alkylcarbonyl, C_1-C_6 alkoxycarbonyl, C_1-C_6 alkoxy, C_1-C_6 alkylthio, arylthio, heterocyclyl, aralkyl, and aryl groups.

23. The method of claim 15, wherein said first agent is a compound or mixture of compounds having the following structure



where Y is $-NR^aR^b$ or $-SO_3^-X^+$, n is an integer from 1 to 5, and X^+ is hydrogen or a cationic group.

24. The method of claim 23, wherein said first agent is a compound or mixture of compounds having one of the following structures

or a pharmaceutically acceptable salt thereof.

25. The method of claim 15, wherein said first agent is 3-amino-1-propanesulfonic acid or a pharmaceutically acceptable salt thereof.

26. The method of claim 1, wherein said first and second agents are administered to said subject together, in a single pharmaceutical composition.

27. The method of claim 1, wherein said first and second agents are administered sequentially.

28. The method of claim 1, wherein at least one of said first and said second agents is orally administered to said subject.

29. The method of claim 1, wherein said second agent is a peptide or peptidomimetic compound that reduces or inhibits amyloid-β fibril formation.

30. The method of claim 29, wherein said peptide comprises hydrophobic amino acids and binds to the hydrophobic region of an amyloid-β peptide, thus blocking β-amyloid fibril formation.

31. The method of claim 30, wherein said peptide comprises one or more modifying groups that enhance the ability of the peptide to block amyloid fibril formation.

32. The method of claim 1, wherein said peptide is an all D peptide.

33. The method of claim 1, wherein said peptide or peptidomimetic is administered to induce a prophylactic or therapeutic immune response against amyloid-β fibril formation, said method further comprising administration of an adjuvant.

34. The method of claim 1, wherein said immune system modulator is selected from the group consisting of antibodies, antibody fragments, T-cells, B-cells, NK cells, NKT cells, dendritic cells, macrophages, basophils, monocytes, and components of the complement pathway.

35. The method of claim 1, further comprising administration of a third agent.

36. The method of claim 35, wherein said third agent is selected from the group consisting of an adrenergic, anti-adrenergic, anti-androgen, anti-anginal, anti-anxiety, anticonvulsant, antidepressant, anti-epileptic, antihyperlipidemic, antihyperlipoproteinemic, antihypertensive, anti-inflammatory, antiobessional, antiparkinsonian, antipsychotic, adrenocortical steroid; adrenocortical suppressant; aldosterone antagonist; amino acid; anabolic steroid; analeptic; androgen; blood glucose regulator; cardioprotectant; cardiovascular; cholinergic agonist or antagonist; cholinesterase deactivator or inhibitor; cognition adjuvant or enhancer; dopaminergic; enzyme inhibitor; estrogen, free oxygen radical scavenger; GABA agonist; glutamate antagonist; hormone; hypocholesterolemic; hypolipidemic; hypotensive; immunizing; immunostimulant; monoamine oxidase inhibitor, neuroprotective; NMDA antagonist; AMPA antagonist, competitive or non-competitive NMDA antagonist; opioid antagonist; potassium channel opener; non-hormonal sterol derivative; post-stroke and post-head trauma treatment; prostaglandin; psychotropic; relaxant; sedative; sedative-hypnotic; selective adenosine antagonist; serotonin antagonist; serotonin inhibitor; selective serotonin uptake inhibitor; serotonin receptor antagonist; sodium and calcium channel blocker; steroid; stimulant; and thyroid hormone or inhibitor agents.

37. The method of claim 1, wherein the concentration of amyloid-β or tau in the cerebrospinal fluid of said subject changes as compared to the concentration in the cerebrospinal fluid of an untreated subject or the treated subject prior to treatment.

38. The method of claim 1, wherein the level of amyloid-β peptides in the plasma of said subject are modulated as compared to the level in the plasma of an untreated subject or the treated subject prior to treatment.

39. The method of claim 1, wherein the level of amyloid-β peptides in the cerebrospinal fluid of said subject are lowered as compared to the level in an untreated subject or the treated subject prior to treatment.

40. A method of preventing or treating an amyloid-β related disease in a subject, said method comprising administering to a subject in need thereof an effective amount of 3-amino-1-propanesulfonic acid, and a second agent that is (i) a peptide or peptidomimetic that modulates amyloid-β fibril formation or induces a prophylactic or therapeutic immune response against amyloid-β fibril formation, or (ii) an immune system modulator that prevents or inhibits amyloid-β fibril formation.

41. A pharmaceutical composition for treating a subject comprising a first agent that prevents or treats amyloid-β related disease, and a second agent that is (i) a peptide or peptidomimetic that modulates amyloid-β fibril formation or induces a prophylactic or therapeutic immune response against amyloid-β fibril formation, or (ii) an immune system modulator that prevents or inhibits amyloid-β fibril formation.

42. The pharmaceutical composition of claim 41, wherein said first agent prevents or inhibits amyloid-β fibril formation, neurodegeneration, or cellular toxicity.

43. The pharmaceutical composition of claim 41, wherein said first agent and said second agent are packaged in separate containers for sale or delivery to consumers.

44. The pharmaceutical composition of claim 41, wherein said first agent and said second agent are dissolved in a liquid pharmaceutically acceptable carrier or are present as a homogenous mixture in a capsule or pill.

45. The pharmaceutical composition of claim 41, further comprising a compound that increases the cerebral bioavailability of either said first agent or said second agent.

46. The pharmaceutical composition of claim 41, wherein said amyloid-β related disease is Alzheimer's disease, mild cognitive impairment, mild-to-moderate cognitive impairment, vascular dementia, cerebral amyloid angiopathy, hereditary cerebral hemorrhage, senile dementia, Down's syndrome, inclusion body myositis, age-related macular degeneration, or a condition associated with Alzheimer's disease.

47. The pharmaceutical composition of claim 46, wherein said Alzheimer's disease is sporadic (non-hereditary) or familial (hereditary).

48. The pharmaceutical composition of claim 46, wherein said condition associated with Alzheimer's disease is selected from the group consisting of hypothyroidism, cerebrovascular disease, cardiovascular disease, memory loss, anxiety, a behavioral dysfunction, a neurological condition, or a psychological condition.

49. The pharmaceutical composition of claim 48, wherein said behavioral dysfunction is apathy, aggression, or incontinence.

50. The pharmaceutical composition of the claim 48, wherein said neurological condition is Huntington's disease, amyotrophic lateral sclerosis, acquired immunodeficiency, Parkinson's disease, aphasia, apraxia, agnosia, Pick disease, dementia with Lewy bodies, altered muscle tone, seizures, sensory loss, visual field deficits, incoordination, gait disturbance, transient ischemic attack or stroke, transient alertness, attention deficit, frequent falls, syncope, neuroleptic sensitivity, normal pressure hydrocephalus, subdural hematoma, brain tumor, posttraumatic brain injury, or posthypoxic damage.

51. The pharmaceutical composition of claim 48, wherein said psychological condition is depression, delusions, illusions, hallucinations, sexual disorders, weight loss, psychosis, a sleep

disturbance, insomnia, behavioral disinhibition, poor insight, suicidal ideation, depressed mood, irritability, anhedonia, social withdrawal, or excessive guilt.

52. The pharmaceutical composition of claim 41, wherein said subject has a genomic mutation in an amyloid precursor protein gene, an ApoE gene, or a presenilin gene.

53. The pharmaceutical composition of claim 41, wherein said subject has amyloid-β deposits.

54. The pharmaceutical composition of claim 41, wherein said subject is a human.

55. The pharmaceutical composition of claim 41, wherein said amyloid-β is an amyloidogenic peptide produced from β-amyloid precursor protein.

56. The pharmaceutical composition of claim 55, wherein said β-amyloid is a peptide having 39-43 amino acids.

57. The pharmaceutical composition of claim 41, wherein said first agent prevents or inhibits β-amyloid fibril formation; prevents β-amyloid peptide, in its soluble, oligomeric form, or in its fibrillar form, from binding or adhering to a cell surface and causing cell damage or toxicity; blocks amyloid-induced cellular toxicity or microglial activation; blocks amyloid-induced neurotoxicity; reduces the rate or amount of β-amyloid aggregation, fibril formation, or deposition; slows the rate of amyloid-β fibril formation or deposition; lessens the degree of amyloid-β deposition; inhibits, reduces, or prevents amyloid-β fibril formation; inhibits amyloid-β induced inflammation; enhances the clearance of amyloid-β from the brain; alters the equilibrium of amyloid-β between the cerebrospinal fluid or brain and the plasma and decreases the amount of amyloid-β in the brain versus the equilibrium distribution in an untreated subject; reverses or favors deposition of amyloid in a subject having amyloid deposits; favors plaque clearance or slows deposition in a subject having amyloid deposits; decreases the amyloid-β concentration in the brain of a subject versus an untreated subject; penetrates into the

brain; maintains soluble amyloid in a non-fibrillar form; increases the rate of clearance of soluble amyloid from the brain of a subject versus an untreated subject; or inhibits or reduces an interation between amyloid-β and a cell surface constituent.

58. The pharmaceutical composition of claim 41, wherein said first agent is a substituted or unsubstituted alkylsulfonic acid, a substituted or unsubstituted alkylsulfuric acid, a substituted or unsubstituted alkylthiosulfonic acid, a substituted or unsubstituted alkylthiosulfuric acid, a substituted or unsubstituted lower alkylsulfonic acid, a (substituted- or unsubstituted-amino)-substituted alkylsulfonic acid, a (substituted- or unsubstituted-amino)-substituted lower alkylsulfonic acid, a substituted or unsubstituted straight-chain alkylsulfonic acid, a substituted or unsubstituted cycloalkylsulfonic acid, a substituted or unsubstituted branched-chain alkylsulfonic acid, or an ester or amide thereof, including pharmaceutically acceptable salts thereof.

59. The pharmaceutical composition of claim 58, wherein said amino substituent has the formula $-NR^aR^b$, wherein R^a and R^b are each independently hydrogen, an alkyl group, an aryl group, or a heterocyclyl group, or R^a and R^b, taken together with the nitrogen atom to which they are attached, form a heterocyclic moiety having from 3 to 8 atoms in the ring.

60. The pharmaceutical composition of claim 59, wherein said heterocyclic moiety comprises a piperidinyl or pyrrolidinyl group.

61. The pharmaceutical composition of claim 58, wherein said amino substituent comprises an alkylamino or dialkylamino group.

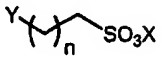
62. The pharmaceutical composition of claim 58, wherein said alkylsulfonic acid comprises an alkyl group substituted with at least a group of the formula $-SO_3H$ or $-SO_3^-X^+$, where X^+ is a cationic group at physiologic pH.

63. The pharmaceutical composition of claim 62, wherein said cationic group is a hydrogen atom, a sodium atom, or an amino group.

64. The pharmaceutical composition of claim 58, wherein said alkylsulfonic acid is substituted with a straight or branched alkyl or cycloalkyl group, or a group of the formula $-NH_2$, $-SO_3H$, $-OSO_3H$, -CN, $-NO_2$, -F, -Cl, -Br, -I, $-CH_2OCH_3$, $-OCH_3$, -SH, $-SCH_3$, -OH, or $-CO_2H$.

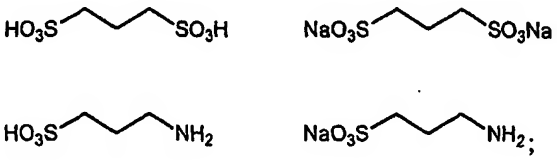
65. The pharmaceutical composition of claim 58, wherein said alkylsulfonic acid is substituted with a substituent selected from the group consisting of halogeno, trifluoromethyl, nitro, cyano, C_1-C_6 alkyl, C_2-C_6 alkenyl, C_2-C_6 alkynyl, C_1-C_6 alkylcarbonyloxy, arylcarbonyloxy, C_1-C_6 alkoxycarbonyloxy, aryloxycarbonyloxy, C_1-C_6 alkylcarbonyl, C_1-C_6 alkoxycarbonyl, C_1-C_6 alkoxy, C_1-C_6 alkylthio, arylthio, heterocyclyl, aralkyl, and aryl groups.

66. The pharmaceutical composition of claim 58, wherein said first agent is a compound or mixture of compounds having the following structure



where Y is $-NR^aR^b$ or $-SO_3^-X^+$, n is an integer from 1 to 5, and X^+ is hydrogen or a cationic group.

67. The pharmaceutical composition of claim 66, wherein said first agent is a compound or mixture of compounds having one of the following structures



or a pharmaceutically acceptable salt thereof.

68. The pharmaceutical composition of claim 58, wherein said first agent is 3-amino-1-propanesulfonic acid or a pharmaceutically acceptable salt thereof.

69. The pharmaceutical composition of claim 58, wherein said second agent is a peptide or peptidomimetic compound that reduces or inhibits amyloid-β fibril formation.

70. The pharmaceutical composition of claim 69, wherein said peptide comprises hydrophobic amino acids and binds to the hydrophobic region of an amyloid-β peptide, thus blocking β-amyloid fibril formation.

71. The pharmaceutical composition of claim 70, wherein said peptide comprises one or more modifying groups that enhance the ability of the peptide to block amyloid fibril formation.

72. The pharmaceutical composition of claim 58, wherein said peptide is an all D peptide.

73. The pharmaceutical composition of claim 58, wherein said peptide or peptidomimetic is administered to induce a prophylactic or therapeutic immune response against amyloid-β fibril formation, and said composition further comprises an adjuvant.

74. The pharmaceutical composition of claim 58, wherein said immune system modulator is selected from the group consisting of antibodies, antibody fragments, T-cells, B-cells, NK cells, NKT cells, dendritic cells, macrophages, basophils, monocytes, and components of the complement pathway.

75. The pharmaceutical composition of claim 58, further comprising a third agent.

76. The pharmaceutical composition of claim 75, wherein said third agent is selected from the group consisting of an adrenergic, anti-adrenergic, anti-androgen, anti-anginal, anti-anxiety, anticonvulsant, antidepressant, anti-epileptic, antihyperlipidemic, antihyperlipoproteinemic, antihypertensive, anti-inflammatory, antiobessional, antiparkinsonian, antipsychotic, adrenocortical steroid; adrenocortical suppressant; aldosterone antagonist; amino acid; anabolic steroid; analeptic; androgen; blood glucose regulator; cardioprotectant;

cardiovascular; cholinergic agonist or antagonist; cholinesterase deactivator or inhibitor; cognition adjuvant or enhancer; dopaminergic; enzyme inhibitor; estrogen, free oxygen radical scavenger; GABA agonist; glutamate antagonist; hormone; hypocholesterolemic; hypolipidemic; hypotensive; immunizing; immunostimulant; monoamine oxidase inhibitor, neuroprotective; NMDA antagonist; AMPA antagonist, competitive or non-competitive NMDA antagonist; opioid antagonist; potassium channel opener; non-hormonal sterol derivative; post-stroke and post-head trauma treatment; prostaglandin; psychotropic; relaxant; sedative; sedative-hypnotic; selective adenosine antagonist; serotonin antagonist; serotonin inhibitor; selective serotonin uptake inhibitor; serotonin receptor antagonist; sodium and calcium channel blocker; steroid; stimulant; and thyroid hormone or inhibitor agents.

77. A pharmaceutical composition comprising an effective amount of 3-amino-1-propanesulfonic acid, and a second agent that is (i) a peptide or peptidomimetic that modulates amyloid-β fibril formation or induces a prophylactic or therapeutic immune response against amyloid-β fibril formation, or (ii) an immune system modulator that prevents or inhibits amyloid-β fibril formation.

78. A kit comprising a first agent that prevents or treats amyloid-β related disease, and a second agent that is (i) a peptide or peptidomimetic that modulates amyloid-β fibril formation or induces a prophylactic or therapeutic immune response against amyloid-β fibril formation, or (ii) an immune system modulator that prevents or inhibits amyloid-β fibril formation.

79. The use of a first agent and a second agent in the preparation of a pharmaceutical composition for the treatment or prevention of an amyloid–β disease, wherein said first agent prevents or treats amyloid-β related disease, and said second agent is (i) a peptide or peptidomimetic that modulates amyloid-β fibril formation or induces a prophylactic or therapeutic immune response against amyloid-β fibril formation, or (ii) an immune system modulator that prevents or inhibits amyloid-β fibril formation.